# PROLAPSE OF THE GASTRIC MUCOSA*

## *Report of Six Cases*

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PROLAPSE OF THE GASTRIC MUCOSA through the pylorus has been considered an unusual phenomenon, and relatively few cases have been reported. The first case was reported by Von Schmieden in 1911.[1] Melamed and Hiller,[2] in a review of the literature in 1943, found only 19 cases reported. Scott[3] reviewed the admissions of a large Naval hospital for the years 1943-1944. In 19,288 admissions, 1,346 examinations of the upper gastro-intestinal tract were made, and in this group 14 instances of prolapse of the gastric mucosa were found, an incidence of 1.04 per cent. In the same series, 13 cases of gastric ulcer were found, an incidence of 0.96 per cent. Rees[4] found only four cases in 3,000 such examinations at the Rees-Stealy Clinic, and only two cases in 2,550 examinations at the San Diego County General Hospital. Archer and Cooper[5] believe that the disease occurs much more frequently than is commonly recognized, but they do not give the incidence. Pendergrass and Andrews[6] report that in the Department of Radiology of the University of Pennsylvania, the condition was diagnosed in 99 patients from 1923 to 1935. They do not, however, give the total number of examinations made during this period.

In a review of the last 97 examinations of the upper gastro-intestinal tract made at the white division of Grady Memorial Hospital, Atlanta, seven cases were diagnosed, an incidence of 7.2 per cent; in the last 100 such examinations in the colored division of Grady Hospital, nine cases were diagnosed, an incidence of 9 per cent; and in 100 gastro-intestinal examinations made at Emory University Hospital, 7 cases were diagnosed, an incidence of 7 per cent. Of the 297 total examinations reviewed at the two hospitals, 23 cases of prolapse of the gastric mucosa were found, a total incidence of 7.7 per cent.[7] Of the 23 cases, four were so extensive as to indicate eventual surgical treatment.

The condition is most often observed in patients in the fourth decade of life, although it has been seen in patients ranging in age from 20 to 80 years. Of the six cases presented here, five occurred in males. The incidence did not vary according to race.

ETIOLOGY. The etiology of prolapse of the gastric musoca is unknown. Eliason and Wright[8] suggest that it is the result of a low-grade inflammation of the mucosa produced by chronic irritation, and that it develops into a local hypertrophy. Once this hypertrophy begins, it is increased mechanically by contraction of the stomach, peristaltic waves, and the pressure of the gastric contents as they are forced by on their way to the pylorus. The mucosa is

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thus pushed along or lengthened out in the direction of the pylorus, and it is eventually swept into the pylorus by a peristaltic wave, causing a ball-valve syndrome attack of pain. Rubin[9] concurs in this theory of an inflammatory origin but believes also that the resulting increase in peristalsis causes a progressive prolapse of the mucosa. Wolf and Wolff[10] found great variation in the thickness of normal mucosa, various stimuli causing large hypertrophic-appearing folds of mucosa which subsequently appeared normal. In three cases of prolapsed gastric mucosa treated surgically, Rees[4] found a "definite resistant narrowing of the pylorus and an apparent loss of muscular substance," suggesting a fibrous degeneration of the muscular tissue. He believes that this narrowing of the pylorus causes hyperperistalsis which in turn loosens the

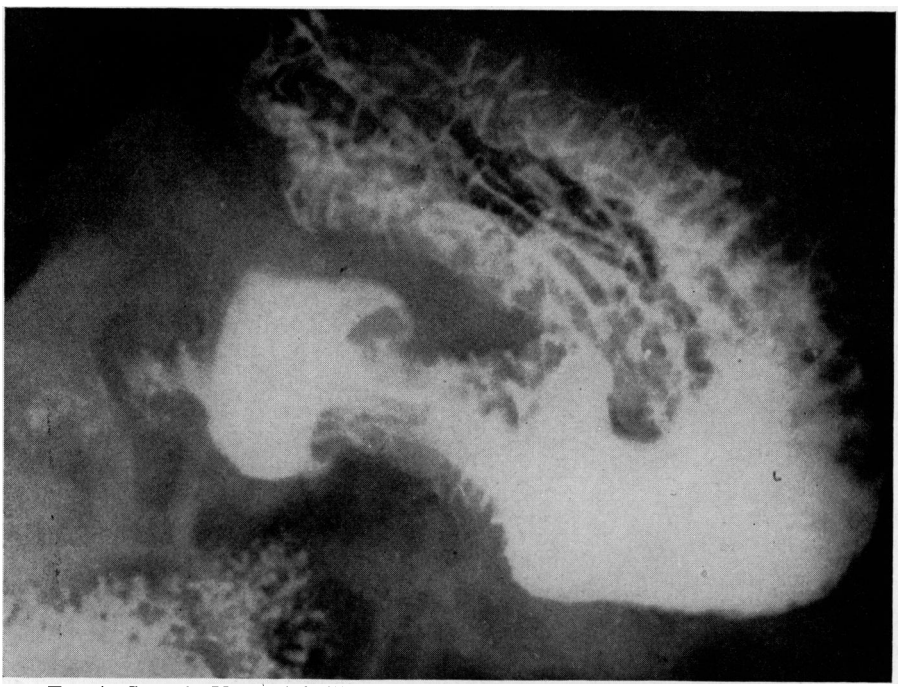

FIG. 1. Case 6: Upper left illustration shows extreme mobility and redundancy of the gastric mucosa. On the right is shown the rugae continuing down well past a normal pyloric aperture into the duodenum.

attachment of the mucous membrane to the muscularis in the constricted area, and that once the membrane is mobilized, the traction is transmitted by continuity to the antral mucosa, which is loosened in turn and eventually prolapses. He states that the condition is progressive and symptoms become more pronounced as the mucosa becomes more redundant. Scott[3] believes that none of these hypotheses will completely explain the condition. Rather, he believes that the mucosa becomes loosened as a result of stretching during normal gastric movements, and he cites Forssell's and Schindler's observations that the mucosa has a mobility of its own, and varies in size, shape

and position independently of the contractions of the muscularis. He states that "the one common denominator in the theories on the etiology of prolapse is an abnormal disturbance of gastric peristalsis and function. The most common continuous and effective means for altering gastric function springs from the emotions and the nervous system. Consequently, in view of the construction of the stomach walls, which normally permits a degree of mobility between them, it seems possible that certain neurogenic factors are the inciting cause of a disturbed gastric function that ultimately brings about a mucosal prolapse. A pre-existing disease process is not necessary for the production of a prolapse of the gastric mucosa."

PATHOLOGY. The pathology involved in prolapse of the gastric mucosa is a redundancy of the mucosa of the pyloric end of the stomach with greatly hypertrophied rugae. This area of mucosa not only becomes redundant, its mobility on the muscularis is greater than normal. Observation at autopsy has shown the mucosa of the normal stomach to be movable on the muscularis, but in no case sufficiently mobile to allow it to be drawn down into the duodenum. In the one case operated upon at Grady Hospital, there was such marked redundancy and mobility of the prepyloric mucosa on the muscularis that the mucosa could be drawn down into the duodenum 6 to 7 cm. beyond the pylorus (Fig. 1). The pyloric muscle was greatly hypertrophied, but the lumen of the pylorus was apparently normal, and the mucosa could be passed in and out of it without difficulty. In this case the prolapsed mucosa could be felt through the duodenal wall, but this was not possible in the majority of cases reported in the literature. The rugae of the stomach were continued on to the prolapsed mucosa. Microscopic examination of the resected mucosa revealed hyperemia and many blood cells throughout but no other evidence of inflammatory change. Some observers have found inflammatory infiltration into the mucosa; [2, 8] others have found a narrowing of the pylorus with loss of muscular substance.[4] The presence of ulcers, polyps, and carcinoma on the prolapsed mucosa has been reported.[9, 2] However, none of these findings have been consistently confirmed by most observers.

SYMPTOMATOLOGY. Pain is the most consistent single symptom and may be either aching or cramp-like in character. It is usually felt in the epigastrium, and frequently radiates either under the costal margin or to the back. It may be prostrating. It occurred in four of the six cases presented here. Nausea and vomiting occurred in five of the six cases. Four of the six had sour belches or heartburn. Four of the patients had hematemesis and melena. Variation of the degree of acidity of the stomach does not appear to be distinctive. Anorexia, anemia (which Archer and Cooper[5] believe has not been adequately stressed as a commonly occurring symptom), and loss of weight may be dominant symptoms. Varying degrees of gastric retention, or none at all, may be present.[4] Thus, it can be seen that these patients present a variable group of symptoms similar to those presented by patients with peptic ulcer.

DIAGNOSIS. There is no definite combination of symptoms common to all cases that will allow the diagnosis to be made from clinical observations alone. Roentgenographic examination is essential in arriving at a diagnosis, and fortunately the findings of this examination are distinctive. There is a filling defect in the duodenum, characterized by a central streak of barium showing an intact mucosa.[4] This defect is variable depending on the degree of prolapse of the mucosa and the motor activity of the stomach at the time

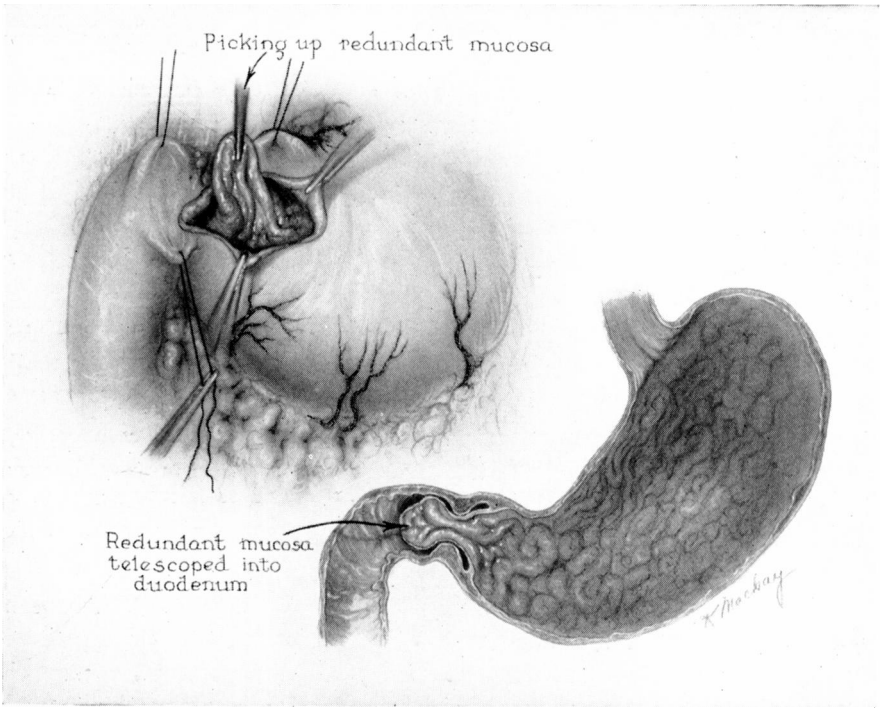


FIG. 2. Case 6: Marked prepyloric deformity with folding out of mucosa into the duodenal cap.

of examination. The mucosa prolapsed into the duodenum allows the barium to flow around it, producing a characteristic mushroom or umbrella effect. Usually the redundant gastric rugae can be traced from the antral canal through the pyloric opening into the base of the duodenal bulb. The rugae in the prepyloric portion of the stomach do not appear abnormal or particularly large.[3] The duodenal bulb is not irritable; it usually retains the barium, thus permitting prolonged examination, a contradistinction to the behavior of the bulb with an active ulcer or duodenitis.[3] Pendergrass and Andrews[6] have summarized the roentgenographic findings as follows:

1. Prolapsing lesions of the gastric mucosa produce a central filling defect in the duodenal cap, and there is a thin shadow of bismuth around the defect.
2. The defect is generally not seen in films made in the erect posture.
3. The diagnosis can be easily overlooked in the fluoroscopic examination.

4. There is no disturbance in the passing of the peristaltic waves.

5. There is always a six-hour residue. (This was not consistently found in the cases observed at Grady Hospital.)

6. There is no defect of the stomach in the presence of pedunculated growths which prolapse through the pyloric ring.

7. There is a variable defect of the prepyloric region of the stomach when prolapsed mucous membrane extends through the pyloric ring.

TREATMENT. The curative treatment of this entity is unquestionably surgical and is directed toward removing the prolapsing mucosa, short circuiting the diseased area, or enlarging the gastric outlet so that the prolapsed mucosa can move back and forth at will without causing obstruction.

Indications for operation are: (1) persistent pain, (2) hemorrhage, and (3) obstruction. Many cases with lesser degrees of prolapse are amenable to dietary management and the administration of antispasmodics. It must be remembered that "the condition is progressive and symptoms become more pronounced as the mucosa becomes more redundant."[4]

Many types of surgical procedures have been used to correct the deformity, including partial gastrectomy, pyloroplasty, gastrojejunostomy, and simple excision of the redundant mucosa. In the one case treated by surgery at Grady Hospital, the redundant mucosa was excised and the appronemated edges were sutured to the muscularis, and a Finney pyloroplasty was done. Probably any procedure that effects removal of the mucosa or gives it freedom to move back and forth without causing obstruction or becoming incarcerated would prove satisfactory.

CASE REPORTS. Summaries of the six cases from the surgical service of Grady Hospital are presented here:

**Case 1.**—E. W., female, Negro, age 23: This patient was admitted June, 1942, complaining of dull aching pain over the left costal margin, more severe when the stomach was empty, not relieved by taking food. She had eructation of sour material, and on three occasions vomited small quantities of blood, the last episode occurring about one week before admission.

Roentgenographic examination revealed prolapse of the gastric mucosa through the pylorus. There was no evidence of ulcer. She was treated with antiacid powder, belladonna, and a fairly liberal diet eliminating greasy foods. There was no further bleeding, and digestive symptoms were relieved. This patient was last seen in 1944, at which time she stated that she was fairly free from digestive symptoms as long as she remained on the diet.

**Case 2.**—L. W., male, Negro, age 39: In January, 1939, this patient vomited dark blood, passed dark, tarry stools, had sudden weakness and blind staggers. He came into the hospital three days later and was treated with Sippy diet and Amphojel for three days. After roentgenologic examination, a diagnosis of hypertrophic gastritis with prolapse of the gastric mucosa was made. He was treated symptomatically, the bleeding stopped, and the patient left the hospital against advice. He was readmitted in September, 1939, and July, 1940, each time following an episode of hemorrhage, and each time the patient left the hospital without permission. In February, 1943, he was admitted to the surgical service because of exsanguinating hemorrhage. Transfusions were given and he recovered

from shock. Roentgenographic examination at that time revealed prolapse of the gastric mucosa into the duodenum. The patient refused surgery and left the hospital.

**Case 3.**—J. A., male, white, age 39: This patient was admitted July 17, 1939, complaining of attacks of epigastric pain, nausea, vomiting, heartburn and indigestion for the previous five years. There was tenderness in the right lower quadrant, and an appendectomy was performed on the day of admission. Pathologic examination showed a normal appendix. Patient left the hospital unimproved. He was readmitted in April, 1945, complaining of having vomited undigested food after meals for a period of ten months. He had never vomited blood. He had had a cholecystectomy at another hospital in January, 1945. Following this operation he had complete relief for three to four weeks, following which he again started vomiting and had almost constant retrosternal pain.

Gastro-intestinal examination showed the pylorus to be somewhat narrowed but the mucosa appeared normal. There was a crescent-shaped filling defect observed in the base of the duodenal cap, representing an hypertrophy of the pylorus and prolapse of the pyloric mucosa into the base of the duodenal cap. Patient left the hospital without permission on April 28, 1945, and was re-admitted in October, 1947, with the same complaints. On a bland diet with belladonna at mealtime he has remained entirely well and has had no further vomiting. This patient had no anemia and vomited no blood at any time.

**Case 4.**—J. L., male, white, age 70: This patient was admitted May 18, 1944, in a coma following subarachnoid hemorrhage. His blood pressure was 190/120. In addition to other difficulties, he had suffered from substernal pain, heartburn, nausea and vomiting over a period of three years, and had passed tarry stools. Gastro-intestinal examination revealed the folds of mucosa in the pylorus to be very thick and to have herniated into the duodenal cap, indicated by a crescent-shaped filling defect in the base of bulb. There was no evidence of ulcer. The patient was placed on a bland diet with antispasmodics. He has had no further discomfort.

**Case 5.**—H. W., male, white, age 49: This patient has had digestive disturbances for the past three years. He has been under considerable emotional strain and has had numerous attacks of substernal pain radiating through to the back. These attacks are relieved by opiates and antispasmodics. For about six weeks following an attack, this patient has all the clinical symptoms of peptic ulcer. A physician, he treats himself over this period with a rather strict diet, between meal feedings, and antispasmodics, and apparently entirely recovers. He is then able to eat normally until the next episode of substernal pain and ulcer symptoms. Repeated roentgenographic examinations have never shown a definite ulcer crater, but there is a definite prolapse of gastric mucosa through the pylorus. It is believed that this prolapse occurs periodically, producing the symptoms of ulcer, but whether actual ulceration occurs on this prolapsed mucosa is a matter of speculation.

**Case 6.**—J. M., male, Negro, age 35: For several years this patient had attacks of abdominal pain beginning in the epigastrium and radiating to the back under the right shoulder blade. These attacks, which lasted two or three hours, were accompanied by heartburn, belching, and occasional vomiting of undigested food. He would take laxatives and recover completely until the next seizure. During an attack July 13, 1947, he vomited about a quart of bright red blood. He continued vomiting until he collapsed. He was admitted to Grady Hospital, July 14, 1947, in a state of shock. His pulse was weak; blood pressure was 80/50; he was sweating and had some air hunger. Hemoglobin was 6.9 Gm.; red blood count was 2 million. He passed several dark, tarry stools. After four transfusions the blood pressure rose to 150/80, hemoglobin to 12 Gm. On July 16, 1947, patient had another episode of bleeding and lost about 1,200 cc. of blood which was replaced by whole blood transfusion. Roentgenographic examination the following day revealed a pronounced prolapse of the gastric mucosa into the duodenal cap, indicated

by an umbrella-like filling defect in the pyloric side of the duodenal cap. (Fig. 2). No other significant changes were noted.

This patient was operated upon August 7, 1947. After the stomach and pylorus were exposed, it was possible to feel the prolapsed mucosa extending for approximately 2.5 cm. into the duodenum. The stomach was opened by a horizontal incision on the anterior surface. The rugae in the prepyloric region of the stomach were very large and were unattached to the muscularis. The mucosa had prolapsed through the pyloric sphincter. With an Allis forcep this loose mucosa could be lifted up in a definite collar-like fold for a distance of 5 or 6 cm. through the incision in the stomach (Fig. 1). The rugae were directed into this collar of mucosa and went on into the duodenum. The collar of mucosa was excised locally and sutured with 00 catgut, taking care to anchor it to the submucous layer. Although the pylorus was patent and appeared normal in size, it was thought wise to transect this muscle. The stomach incision was therefore extended on to the duodenum and a classic Finney pyloroplasty was done. No evidence of ulcer was found. The site of the bleeding was not located. The mucosa was of normal color and appearance.

The patient became ambulatory the following day. Roentgenographic examination showed no evidence of further prolapse, but an enlarged, rather deformed-looking pylorus now exists. The patient had no further symptoms and was discharged from the hospital August 19, 1947. He was last seen November 25, 1947 and had been entirely symptom free since operation.

## SUMMARY

1. Prolapse of the gastric mucosa through the pylorus is a distinct clinical entity.

2. This condition occurs rather frequently, being found more often than gastric ulcer. In a review of the last 297 examinations of the upper gastrointestinal tract made at Grady Memorial and Emory University Hospitals, 23 cases were found, an incidence of 7.7 per cent.

3. The diagnosis may be easily missed. This disease may well be the underlying factor in many of the undiagnosed digestive disturbances.

4. The etiology is unknown.

5. The pathology is an abnormal mobility and redundancy of the prepyloric mucosa with prolapse through the pylorus.

6. The symptomatology is not clearly defined.

7. Roentgenographic examination is essential in making the diagnosis. The findings are distinctive.

8. The curative treatment is surgical, but milder cases can be successfully treated by dietary management and antispasmodics.

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DISCUSSION.—DR. T. C. DAVISON, Atlanta, Ga.: I would like to ask what produces the symptoms. Is it obstruction in the pylorus?

DR. IRA A. FERGUSON, Atlanta, Ga. (closing): We thought the symptom of pain was probably produced by spasm of the pyloric sphincter. There is, of course, a question as to whether the prolapsed mucosa becomes strangulated or so incarcerated in the pyloric aperture that it interferes with the blood supply. We think this is a possibility as hemorrhage is a commonplace symptom. However, the most likely cause is pylorospasm.